Nicotine and cardiovascular disease

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Cigarette smoking is a major risk factor for coronary and peripheral vascular disease. This paper addresses the importance of nicotine per se in causation of heart disease. In people with coronary artery disease, nicotine, by increasing the work and oxygen demand of the heart, can induce ischemia that may contribute to reduced exercise capacity and/or impaired myocardial contractility. Coronary spasm and enhanced thrombosis are believed to contribute to smoking-related myocardial infarction. Nicotine may contribute to coronary spasm by sympathomimetic actions or inhibition of local release of prostacyclin. The role of nicotine in causing hypercoagulability has not been established. Nicotine could promote atherosclerotic vascular disease by actions on lipid metabolism and coagulation, by hemodynamic effects and/or by causing endothelial injury. Nicotine feeding has been shown to increase LDL cholesterol in some animal studies, but such effects have not been confirmed in most studies of humans chewing nicotine gum. Nicotine produces structural damage to aortic endothelial cells in animals, but the significance of this effect to human disease is unclear. Arrhythmias could contribute to sudden death in smokers. Cigarette smoking does not increase the prevalence of ventricular ectopy in people with stable ischemic heart disease. However, nicotine facilitates AV nodal conduction and may render arrhythmias that occur due to acute ischemia more life-threatening. In summary, nicotine may contribute to tobacco-related cardiovascular disease, but direct causation has not been determind. Short-term nicotine therapy, such as nicotine replacement therapy as an adjunct to smoking cessation therapy, poses little cardiovascular risk to healthy individuals.

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